"STUDY TO EVALUATE CLINICAL PROFILE OF SEIZURES IN CASES OF STROKE"

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CERTIFICATE

This is to certify that the dissertation titled **"STUDY TO EVALUATE CLINICAL PROFILE OF SEIZURES IN CASES OF STROKE"** is the bonafide work of DR.C.S.Selvarathinasamy in partial fulfillment of the requirements for the D.M., Branch I (Neurology) examination of THE TAMILNADU DR M.G.R.MEDICAL UNIVERSITY to be held in August 2011.

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DECLERATION

I **Dr. C.S. Selvarathinasamy,** hereby declare that the present study, entitled **"STUDY TO EVALUATE THE CLINICAL PROFILE OF SEIZURES IN CASES OF STROKE"** is the bonafide work done by me at Government Stanley Medical College Hospital under the guidance and supervision of **Prof. S.GOBINATHAN, M.D., D.M.,** Professor and Head of the Department of Neurology. This dissertation is submitted to The Tamilnadu Dr M.G.R. Medical University, towards partial fulfillment of requirement for the award of D.M. Degree examination to be held in August 2011.

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CONTENTS

INTRODUCTION

Stroke is defined as sudden onset of neurological dysfunction secondary to vascular etiology either ischemic or hemorrhagic. Stroke is associated with a significant mortality and high morbidity. It is the third leading cause of death in developed countries and in developing countries it is fast catching up as one of the leading causes of death.

Seizures is a common neurological disorder. John Hughling Jackson first recognized the relation between stroke and seizures more than a century ago stroke has been considered one of the most common causes of epilepsy in adult population especially in people above 60 years age. Post stroke seizures significantly increase morbidity in stroke patients. Seizures occur in about 8-10% of stroke patients. About 10% of patients with post stroke seizures present with status epilepticus. Seizures after stroke are common but development of epilepsy is rare.

Post stroke seizures are divided into early (onset within two weeks) and late (onset after two weeks). Early onset seizures are considered to be secondary to local metabolic and toxic factors, while late onset seizures occurs secondary to gliotic area following stroke. Chances of epilepsy are more after late onset seizures.

Incidence of post stroke seizures is more in hemorrhagic stroke than in ischemic stroke. Cortical lesions are more likely to cause seizures than sub

cortical and larger lesions are more likely to cause seizures. Venous infarcts cause seizures more frequently than arterial infarcts.

Post stroke seizures adversely affect the outcome of stroke. Post Stroke seizures usually require short term antiepileptic drug treatment and in most instances are well controlled by monotherapy. Still there is no consensus regarding prophylactic antiepileptic therapy, but American Heart Association recommends prophylactic antiepileptic drug treatment in cases of lobar hematoma and sub arachnoid hemorrhage.

Most of the studies of post stroke seizures are retrospective analysis of case records. There have been very few prospective studies to define the incidence of seizures in stroke and their clinical profile.

The present study was done to evaluate the clinical profile on post stroke seizures prospectively in south Indian Population.

REVIEW OF LITERATURE

Stroke is associated with high mortality and morbidity. It is the third leading cause of death in developed countries. There are no reliable epidemiological data on stroke applicable to whole of the India. Very few population-based studies are available. Banerjee Tk etal[1] in Calcutta conducted a Population-based cluster survey on stroke disorders. The crude prevalence rate of stroke was 147/1,00,000 (age adjusted prevalence 334/1,00,000). The annual incidence rate of stroke for the year 1998-1999 was 36/1,00,000. There was relatively more cases of cerebral hemorrhage in survey, compared to those in the western countries. Hypertension was the most important rise factor in this study.

Seizures usually occur in about 8-10% of stroke patients[2], but in literature reported frequency ranges from 08-17%. seizures after stroke are common but development of epilepsy is rare.

Post stroke seizures are divided in to early (onset within two weeks of stroke), and late (onsent after two weeks of stroke[2,3]. Early onset seizures are considered to be secondary to local metabolic and toxic factors, while late onset seizures occur secondary to gliotic area following stroke. Chances of epilepsy are more after late onset seizures.

Incidence of post stroke seizures is more in hemorrhagic stroke than in ischemic stroke. Cortical lesions are more likely to cause seizures than

subcortical and larger lesions are more likely to cause seizures. Venous infarcts cause seizures more frequently than arterial infarcts[2,3.]

The largest and most rigorous methodological attempt examine post stroke seizures was the prospective multi center study 'seizure after stroke study group' by Bladin CF etal [4].

They studied the incidence, outcome, and risk factors for seizures after stroke in 2021 consecutive stroke patients. After exclusion of 124 patients with previous epilepsy or without computed tomographic diagnosis, 1897 were analysed. Seizures occured in 168(8.9%) of 1897 patients with stroke, 28(10.6%) of 265 with hemorrhagic and 140 (8.6%) of 1632 with ischemic stroke. Patients with hemorrhagic stroke were at significantly greater risk of seizures (p=0.002). Risk factors for seizures after ischemic stroke were cortical location of infarction and stroke disability. The only risk factor for seizures after hemorrhagic stroke was cortical location. Recurrent seizures (epilepsy) occurred in 47 (2.5%) of 1897 patients. Late onset of the first seizures was an independent risk factor for epilepsy after ischemic stroke but not after hemorrhagic stroke.

Hauser WA etal[5] studied the incidence of epilepsy and all unprovoked seizures for residents of Rochester, U.S.A. from 1935 through 1984. Age adjusted incidence of epilepsy was 44 per 1,00,000 person – years. Incidence in males was significantly higher than in females and was high in the first year of

life. The highest incidence was noted in persons aged 75 years or more. Sixty percent of new cases had epilepsy manifested by partial seizures. Two third patients had no clearly identified antecedent event. Cerebrovascular disease was the most commonly identified antecedent, accounting for 11% of cases.

Sung CY et al[6] reported that among 1402 patients with intracerebral hemorrhage (ICH), seizures occurred in 64 (4.6%) and epilepsy in 35 (2.5%). Seizures was the first manifestation of ICH in 19 patients (30%). Status epilepticus occurred in 11 patients (17%) and it was the initial presentation of ICH in six (9%). The majority had simple partial seizures that were predominantly focal and motor. There were 38 patients with early seizures and 26 patients with late seizures. Ninety percent of seizures occurred within one year after ICH. Eleven patients (29%) with early seizures developed epilepsy, where as 24% patients (93%) with late seizure developed recurrent seizures. The incidence of seizure was 32% for lobar hematoma, 2% respectively for putaminal, thalamic and pontine hemorrhages and 1% for cerebellar hemorrhage. 26 out of 42 (62%) patients with lobar hematoma developed epilepsy. Thirteen patients (34%) with early seizures died within three months after the onset of seizures whereas three patients (12%) with late seizure died within the same period.

Burn J et al[7] described the immediate and long term risk of epileptic seizures after first ever stroke in community based stroke study. 675 patients with a first stroke were followed up for a minimum of 2 years. Fifty two

patients had one or more post stroke seizures, in 25 patients the seizures were recurrent. The 5 year risk of post stroke seizure in survivors was 11%. The relative risk of seizures, in comparison with the general population was estimated at 35.2 in the first year after stroke and 19.0 in the second year. The risk of seizures was increased in survivors of subarachnoid hemorrhage and intra cerebral hemorrhage. The risk of seizures after ischemic stroke was substantial only in patients presenting with severe stroke due to total anterior circulation infraction. Stroke patients have about 11.5% risk of single or recurrent seizures in the first 5 year after a stroke. Patients with more severe strokes or hemorrhagic strokes are at higher risk.

Gupta SR et al[8] retrospectively studied 90 patients with post infarction seizures 33% seizures appeared early (within 2 weeks) and 90% of the 30 early seizures appeared within 24 hours. 73% of the 90 seizures occurred within the first year. Fifty six percent of the 90 seizures were single, and status epilepticus was seen in only 8%. Early onset seizures were more likely to be partial (57% of 30%); late onset seizures were more likely to be generalized (65% of 60). Thirty nine percent of the 90 initial seizures recurred, and there was no significant difference in recurrence rate between early or late onset initial seizures. Twenty-two percent of the 90 initial seizures became multiple recurrent seizures, and authors could identify a precipitating factor in 86% of the 35 recurrent seizures. The most common EEG abnormality was focal slowing (61%), but recurrent seizures occurred in 100% of four patients with

periodic lateralized epileptiform discharges and in 75% of the eight patients with diffuse slowing. CT scans in 61 patients showed that large infarctions were associated with early and multiple seizures. Deep infarctions on CT scan (cortical infarctions extending to subcortical structures) tended to cause recurrent seizures.

Giroud M et al[9] prospectively evaluated the occurrence of seizures within 15 days of a first stroke or transient ischemic attacks in 1,640 patients to study the relation between seizures and the type of stroke. Seizures occurred in 90 patients (5.4%) including 36 (4.4%) of 814 with infarct due to atheroma, 21 (16.6%) of 126 with infarct due to cardiogenic embolus, 3 (1%) of 273 due to lacunar infarct, 5 (1.9%) of 259 due to transient ischemic attack (TIA), 21 (16.2%) of 129 due to supratentorial hematoma, and 4 (16.6%) of 24 due to subarachnoid hemorrhage. Thirteen (14.6%) of 89 sub cortical infarcts were associated with seizures. Seizures were the initial sign of stroke in 80 (89%) of 90 cases and were usually single and partial.

Kilpatrick CJ et al[10] assessed the incidence of late seizures in 31 patients with early seizures complicating acute stroke and compared this with the incidence of late seizures in 31 matched patients with stroke without early seizures. Ten (3.2%) of 31 patients with early seizures had late seizures during a mean follow-up period of 26 months. Only three (10%) of 31 patients without early seizures had late seizures during the follow-up period of 28 months, a significantly lower incidence than in patients with early seizures. The risk of

seizure recurrence in patients with early seizures did not correlate with stroke type or lesion size as imaged on the computed tomographic scan. According to the authors early seizures are not benign and are associated with a significant risk of seizure recurrence.

Kotila M etal[11] retrospectively followed 200 stroke patients (ischemic brain infarction (IBI) 157, intracerebral hemorrhage (ICH) 20, and SAH 23) for a mean period of 40 months after stroke. Epilepsy developed in 33 (17%) patients. The occurrence of epilepsy was 14% in IBI, 15% in ICH, and 35% in SAH. Significantly more patients developed epilepsy in the SAH group than in the IBI group (8 of 23 vs 22 of 157, $p<0.05$). Of the 33 patients, 15% had their first seizures within the first 2 weeks after stroke, and 55% developed epilepsy in 6 months. Forty-eight percent of the patients had generalized seizures.

Reith J et al[12] in their study determined the relationships between epileptic seizures and stroke outcome and identified predictors of epileptic seizures. In community bases study, they prospectively and consecutively studied 1197 patients with acute stroke. Fifty patients (4.2%) had seizures within 14 days of stroke. In multivariate analyses, only initial stroke severity was related to epileptic seizures; stroke type and lesion localization were not related. For each 10-points increase in stroke severity, the relative risk of epileptic seizures increased by a factor of 1.65 ($p<0.0001$). Early onset seizures did not influence the risk of death during hospital stay ($p=0.56$). The decisive factor of early onset seizures was initial stroke severity. Early onset seizures

per se were not related to mortality. Surprisingly early onset seizures predicted a better outcome. The authors explained this finding by relatively larger ischaemic penumbra in patients who have epileptic seizures after a stroke.

Rumbach L et al[13] evaluated the incidence, clinical data, and prognostic factors of status epilepticus after stroke. They included 3,205 patients admitted with first time strokes from 1984 to 1994. First time post stroke seizures were noted in 159 patients. Status epilepticus was recognized in 31 patients (19%). In 17 patients status epilepticus was the first epileptic symptom (initial status epilepticus), and in 4 patients, stroke began with status epilepticus. In the remaining 14 patients status epilepticus occurred after one or more seizure(s). After a mean follow-up period of 47 months, neurological deterioration occurred after status epilepticus in 15 patients. This deterioration was permanent in two patients. Fifteen died; in five patients, death was directly related to status epilepticus. Recurrent seizures or status epilepticus was seen in 8 out of 17 patients with initial status epilepticus and all 14 patients with status epilepticus after one or more seizure(s).

Shinton RA et al[14] assessed the frequency and significance of epileptic seizures at onset of stroke in 230 patients. Thirteen (5.7%) patients suffered single or multiple seizures at the onset of their stroke. Seizures were evenly distributed among all pathological stroke sub-types but were restricted to lesions in the carotid artery territory. They indicated a poorer prognosis over the first 2 days. Six of the 13 presenting with stroke and seizures had prior

seizures and, if they survived, continued to have fits. The five patients surviving with stroke and a first seizure were all seizure free after 30 months follow-up.

Lancman ME et al[15] evaluated the development of seizures in 219 consecutive stroke patients. Twenty two of 219 stroke patients (10.04%) had seizures after stroke. Twelve (54.55%) were of early onset (<1 month), and 10(45.45%) were of late onset. No statistically significant differences were evident between the early-and late-onset seizure group in comparisons of type of stroke, localization, and the size of lesion. Six of 22 patients (27%) had seizure recurrence. Seizures developed in (a) 13 of 183 patients with ischaemic stroke (7.1%) and 9 of 36 patients with hemorrhagic stroke (25%) (p=0.01); (b) 16 of 93 patients with cortical lesions 17% and 6 of 126 patients with sub cortical lesions (4.7%) (p=0.01); and (c) 14 of 66 patients with a lesion comprising more than lobe (21.2%) and 8 of 153 patients with a lesion comprising less than one lobe (5.2%) (p<0.01). Patients with hemorrhagic stroke, cortical lesion, and lesions involving more than one lobe were at higher risk of developing seizures.

G.Devuyst et al[16] studies the role of serum cholestrol in prodromal and early epileptic seizures in acute stroke. Patients presenting seizures <1 week before (n=6), 3 hours after (n=26) and 3 to 24 hours after (n=11) a first ever stroke were included. Seizure patients had lower levels of serum cholestrol on multivariate analysis (p<0.0001). Mortality and functional out come at

discharge were not influenced by serum cholestrol. Early post stroke seizures occur mainly during the critical 3 hour window for thrombolysis. Hypercholesterolemia appears to protect against seizures and cerebral ischemia.

Arboix A et al[17] studied the influence of early post stroke seizures (within 48 hours of stroke or TIA) on mortality in 1,099 consecutive patients. 27 Patient (2.5%) had epileptic seizures during the first 48 hours of stroke. Advancing age, confusional syndrome, hemorrhagic stroke, large lesions, involvement of parietal and temporal lobes, and occurrence of neurologic and medical complications were significantly more frequent in seizure patients than in non seizure patients. Over all in-hospital mortality rate was 33.3% in the seizure group and 14.2% in the non seizure group (p=0.02). Seizures at the onset of a first ever stroke were an independent prognostic factor for in-hospital mortality. Patients with the highest risk of developing epileptic seizures were aged persons with a large hemorrhagic infarction of a parietal lobe. These patients may be candidates to be treated prophylactically with antiepileptic drugs for a few days.

Arboix A et al[18] studied the predictive factors of early seizures in first ever stroke patients. Data of 1220 first ever stroke patients admitted between January 1986 and December 1993 were collected. Early epileptic seizures were diagnosed in 29 (2.4%) patients. Seizures were significantly more frequent in patients with hemorrhagic stroke (4.3%) than in those with ischaemic stroke

(2%) patients with seizures were significantly younger and more likely to have acute confusional state, cortical involvement, large stroke, and involvement of the parietal, frontal, occipital, and temporal lobes than patients without seizures. The in-hospital mortality rate was 37.9% in the seizure group and 14.4% the non seizure group ($p<0.0005$). After multivariate analysis, only cortical involvement (odds ratio of 6.01) and acute agitated confusional state were independent clinical factors for developing epileptic seizures.

Paolucci S et al[19] designed a study to identify the prevalence of post stroke late seizures in a population of patients admitted to rehabilitation of neurologic sequelae of their first stroke, recognize reliable prognostic factors associated with the occurrence of post stroke late seizures, and evaluate the impact of seizures on the results of rehabilitation treatment. This was a prospective study of 306 consecutive stroke patients. They also evaluated the impact of post stroke seizures on both efficiency and effectiveness of rehabilitation and length of stay. Post stroke late seizures occurred in 46(15.03%) patients, with a mean interval from stroke of 101.98 ± 37.96 days. In multiple regression analysis, putaminal and lobar hemorrhages showed a significant positive association with the development of seizures ($p<0.005$), whereas high scores on the canadian neurological scale and increasing age were negatively associated ($p<0.01$ and $P<0.05$). Patients with putaminal and lobar hemorrhages and patients with severe stroke were significantly greater

relative risk of seizures. No Significant association was found between post stroke seizures and results of rehabilitation.

GD Baquis et al[20] described eight patients with an unusual form of carotid transient ischemic attack, limb shaking. The basic features included a brief, involuntary, coarse, irregular, wavering movement or tremble involving arm-hand alone or arm-hand and leg together. In 2 patients limb shaking was the initial manifestation of carotid occlusive disease, and all but one patient had other typical carotid transient ischemic attacks. Major atheromatous carotid occlusive disease was present in all patients on the side opposite the limb movements. Four patients had bilateral carotid occlusive disease. Cerebral ischemia from a carotid territory low-perfusion state may be the pathogenesis of these limb movements, an idea supported by the apparent benefit of surgical revascularization in abolishing or reducing the limb shaking in 6 patients. There was no clinical or EEG evidence to document an epileptiform etiology.

Bogousslavsky J et al[21] reported the effect of seizure on stroke sequelae. They described 10 patients with post stroke partial epileptic seizures that were followed by persistent worsening of previous neuralgic deficit. Of 38 other patients with post stroke seizures who were examined during the same period, eight suffered transient neurologic worsening (Todd's phenomenon). Persistent worsening was associated with longer seizures and longer partial seizures before generalization. Risk factors, age, sex, other seizure features, and characteristics of previous stroke were irrelevant to developing persistent

worsening of stroke sequelae. None of the patients with persistent worsening showed a new lesion or an extension of the previous ischemic area on computed tomography or magnetic resonance imaging, except one who had a first hemorrhage that spared the cortex and who suffered a second hemorrhage, which was lobar. Persistent worsening of a neurologic deficit following a seizure may be due to a direct effect of the seizure itself on the infarcted area.

Cocito L et al[22] investigated the occurrence of epileptic seizures in 141 patients with angiographically proven carotid or middle cerebral artery (MCA) occlusive disease. Epileptic seizures occurred sometime during the clinical course of the disease 17.3% of carotid patients and in 10.8% of MCA patients. Partial motor seizures were more common. The pattern of occurrence of seizures in the natural history of cerebral arterial disease was different in the two groups. In the carotid group, epilepsy was the presenting symptom in 6.7% of patients, whereas no MCA patients had seizures prior to the appearance of the neurological deficit.

Giroud M et al[23] in a population-based study evaluated, seizures occurring in the first 15 days after strokes among 1,640 consecutive patients who had ischaemic or hemorrhagic stroke on CT scan. Ninety patients had an epileptic seizure in the first 15 days after stroke onset. Thirteen of the 90 had a lenticulostriate infarct, diagnosed on CT scan, without an apparent ipsilateral cortical ischemic lesion. No lenticulostriate hematoma was observed with seizures. To determine the possible existence of an ipsilateral cortical lesion,

MRI with gadolinium perfusion, and single photon emission CT (SPECT) were performed in the 13 patients with seizures. MRI showed an associated ipsilateral posterior frontal or anterior temporal cortical ischaemic lesion in 11 cases, and SPECT showed decreased blood flow in the ipsilateral frontal area in all cases(superficial sylvian territory). Overall, 56 patients had a lenticulostriate infarct and clinical, CT, and MRI date from the 13 with seizures was compared with those of the 43 without seizures. Two criteria differentiated the two groups: the size of lenticulostriate infarct was larger (8.3 vs. 3.9 cm 3) and ipsilateral cortical ischemic lesions were more frequent in the group with seizures (84 vs. 9%)

Heuts-van REP et al[24] studied the frequency of supratentorial brain infarction as the cause of late onset epilepsy in 680 patients with a first seizure after the age of 20 years. 65 (10%) had seizures following a symptomatic supratentorial brain infarct. First seizure occurred within one year after brain infarction in 62%, and after two years in 19%. Eight of 14 patients (57%) with an early seizure (2 weeks or less), and 28 of 41 patients (68%) with a first seizure later than two weeks post-stroke had seizure recurrences despite anti-epileptic treatment. Of 38 patients who underwent computed tomography scan (CT), 32 (84%) had a cortical infarct, where as six (16%) had one or more lacunar infarcts. Lacunar infarction may be associated with post-stroke epilepsy. Using a detailed topographic brain atlas to localize the cortical infarcts, no specific epileptogenic gyri could be identified.

Heutz-van REP et al[25] studied prospectively the occurrence of seizures in 322 patients with a first-ever CT-confirmed symptomatic territorial brain infarct involving the cortex. They also studied potential risk factors for seizures, and with special attention to cortical infarct location. Fifty-four patients developed post stroke seizures. Patients older than 65 years with a cardio embolic brain infarct involving the middle temporal or post central gyrus, had an almost eight times increased risk of early onset seizures. Whereas patients with a large brain infarct involving the supramarginal or superior temporal gyrus, had a five times increased risk of late onset seizures. Authors reported that risk factors and epileptogenic cortical areas for post brain infarct seizures could be identified, which however, differ between early-and late onset seizures.

Kilpatrick CJ et al[26] evaluated the incidence of early seizures in 1000 patients with stroke and transient ischaemic attacks prospectively. Seizures occurred in 44 patients (4.4%), status epilepticus (0.7%), including 10(15.4%) of 65 with lobar or extensive hemorrhage, 6(8.5%) of 71 with subarachnoid hemorrhage, 24 (6.5%) of 370 with cortical infarction and 4 (3.7%) of 109 with hemispheric transient ischaemic attacks. Lacunar infarct and deep hemorrhages were not associated with seizures. Arteriovenous malformation was a common cause of lobar hemorrhage with early seizures, but in cortical infarcts there was no association between seizure occurrence and pathogenesis. Seizures generally occurred within 48 hours of stroke onset, were usually single, partial,

and readily controlled. Seizures were not associated with a higher mortality or worse functional out come.

Labovitz DL et al[27] studied the prevalence and predictors of early seizure and status epilepticus after first stroke in adult residents of northern Manhattan the cohort consisted of 904 patients; early onset seizures occurred in 37 (4.1%). The frequency of early onset seizures by stroke sub type and location was deep infarct 0.6% (2/356), lobar infarct 5.9%) (20/341), deep intracerebral hemorrhage (ICH) 4.0% (4/101), lobar ICH 14.3% (7/49), and subarachnoid hemorrhage 8.0% (4/50) status epilepticus occurred in 10 patients (1.1%), representing 27.0% of patients with early onset seizures. Diabetes, hypertension, current smoking, alcohol use, age, gender, and race/ethnicity were not significant determinants of epileptic seizures. NIH stroke scale score was not an independent predictor of early onset seizures in multivariate analysis. After accounting for stroke severity, early seizures were not a predictor of 30 day case fatality. Lesion location and stroke subtype are strong determinants of early onset seizures risk, even after adjusting for stroke severity. Early onset seizures do not predict 30 day mortality. Status epilepticus occurs in more than one quarter of patients with early onset seizures.

Sitajayalakshmi S et al[28] in their review of post stroke seizures reported that early onset seizures occur within two weeks of stroke onset, while late onset seizures occur after two weeks. The incidence of early seizures is high with lobar hemorrhage, cortical infarcts especially embolic, agitated acute

confusional state and increased stroke severity at stroke onset. Both early and late onset post stroke seizures left sided cortical infarcts, increased stroke severity and recurrent strokes are the risk factors for post stroke late epilepsy. Post stroke early seizures as well as late epilepsy do not significantly affect long term outcome and rehabilitation of stroke.

So EL et al[29] performed the population based study to determined the magnitude of the risk and identify the factors predictive of developing seizure disorders after cerebral infarction. 535 consecutive patients without prior unprovoked seizures were followed from their first cerebral infarctions until death or migration out of Rochester, Minnesota. Thirty-three patients (6%) developed early seizures (within 1 week), 78% of which occurred within the first 24 hours. Using multivariate analysis, the only factor predictive of early seizure occurrence was anterior hemisphere location of infarct. 27 patients developed an initial late seizure (past 1 week), whereas 18 developed epilepsy. Compared with the population in the community, the risk during the first year was 23 times higher for initial late seizures and 17 times higher for epilepsy. The cumulative probability of developing initial late seizures was 3.0% by one year, 4.7% by 10 years. Independent predictive factors on multivariate analysis for initial late seizures were early seizure occurrence (hazard ratio of 7.8) and stroke recurrence (3.1). Both early seizure (16.4) and stroke recurrence (3.5) independently predicted the development of epilepsy as well.

Weisberg LA et al[30] studies the occurrence of seizures in non traumatic parenchymal brain hemorrhages. Seizures occurred in 15% of patients with parenchymal brain hemorrhage (early in 12% and delayed in 3%). Seizures were more frequent with lobar hemorrhages and uncommon with deep subcortical hemorrhages. Lobar hemorrhages in the frontal, parietal, or temporal region were more commonly associated with seizures, whereas occipital hemorrhages were not. Seizures were more common if the hemorrhage was due to an aneurysm, angioma, or neoplasm and less common if hypertensive or spontaneous. If the patient had recurrent seizures or developed delayed seizures, CT showed that hemorrhage evolved to a hypodense appearance. CT showed that the hemorrhage evolved to an isodense appearance in patients without seizure recurrence.

Various EEG changes have been described in cases of stroke. These include diffuse background showing, hemispheric slowing, and periodic lateralized epileptiform discharges (PLEDS) ipsilateral to the site of lesion. Also, bilateral independent periodic transients (BIPLEDS) can occur in strokes complicated by hypoxia and/or infection. EEG can also be used to predict the chances of having seizures after stroke, as well as in cases of carotid endarterectomy.

Pohlman EB et al[31] reviewed the literature dealing with electroencephalographic (EEG) pattern periodic lateralized epileptiform discharges (PLEDS). Functional neuroimaging studies, strongly suggests that PLEDS might reflect a key pattern for focal hyper excitability in the penumbra zone of ischemic stroke. They considered PLEDS as an EEG signature of a dynamic pathophysiological state in which unstable neurobiological processes create an ictal - interictal continuum, with the nature of the underlying neuronal injury, the patient's pre-existing propensity to have seizures, and the co-existence of any acute metabolic derangements all contributing to whether seizures occur or not.

Verma NP et al[32] described the contralateral epileptiform transients (CETS) in EEG in cases of stroke. This phenomenon is apparently rare and consists of non periodic spikes occurring singly or in bursts, sharp waves, or sharp-and-slow waves occurring contra laterally to an acute stroke. These discharges are not associated with clinical seizures and appear to last longer than PLEDS or BIPLEDS. Sometimes, they may point to a contralateral cerebral and/or cardiac pathology.

Gilmore PC[33] et al studied the correlation of EEG, computerized tomography, and clinical findings in 100 patients with focal delta activity on EEG. Sixty eight percent showed focal structural lesions on CT, with stroke being the most frequent etiologic factor followed by tumors. Convulsions were the most frequent cause of focal delta activity with a normal scan.

Alvarez-Sabin J et al[34] evaluated the long term efficacy and tolerability of gabapentin (900 to 1,800 mg/day) in 71 patients with a first post stroke late ES during a mean follow-up time of 30 months. Epileptic seizures recurred in 18.3% of the patients and side effects were noted in 27 cases (38%), but only two (2.8%) required discontinuation or early withdrawal. Gabapentin monotherapy was useful and safe for late post stroke epileptic seizures.

Roberts RC et al[35] reported the clinically unsuspected cerebral infarction in late onset epilepsy. The CT scans of 132 patients with late onset epilepsy were compared with the CT scans of an age and sex matched control group. Fifteen of the patients with epilepsy, as opposed to two of the controls, had infarcts on their CT scans ($p=0.003$). In nine of these patients only lacunar infarcts were present. No patient had a history of stroke. Twelve of the 15 patients were aged greater than 60 years, representing 21% of the patients in this age group. There was no difference between the epileptic patients and controls in the presence of clinical features of systemic vascular and cardiac disease.

Velioglu S et al[36] evaluated the profile of status epilepticus after stroke. From 1988 of 2000, 1174 patients were admitted to the department of neurology at the Karadeniz Technical University with first time strokes. 180 patients had post stroke first-time seizures. These patients were followed for an average of 3.7 years or until death to determine the occurrence rate of status epilepticus. Total of 17 of 180 post stroke seizure patients (9%) had status

epilepticus. There was no relationship between the occurrence of status epilepticus and stroke risk factors, stroke type, stroke topography and cause, cortical involvement, size of lesion, seizure type, or electroencephalographic findings. Status epilepticus occurred more frequently among patients with a higher disability rating (Rankin Scale >3.) Recurrent status epilepticus was identified in 5 of 17 patients with status epilepticus. In all 5 patients the first episode of status epilepticus occurred within the first 7 days after stroke (early-onset status epilepticus). Statistical analysis demonstrated that early onset status epilepticus was associated with a higher risk for recurrence ($p=0.003$) and a higher mortality rate ($p=0.04$). Status epilepticus was not associated with a higher mortality rate but with higher functional disability.

Seizures are well documented in other vascular disorders like subarachnoid hemorrhage, arterio-venous malformation and cortical venous thrombosis. Prophylactic anticonvulsant treatment is usually recommended in cases of subarachnoid hemorrhage and cortical venous thrombosis. In cases of arterial infarcts there are no well documented trials of usefulness of prophylactic anticonvulsant therapy. However American Heart Association (AHA) recommends use of prophylactic anticonvulsant in large parenchymal hematoma. Antiepileptic drugs can be discontinued after one month if there are no seizures.

AIMS AND OBJECTIVES

(1) To evaluate the incidence of post stroke seizures.

(2) To evaluate the type and latency of seizure after stroke and clinical variables.

(3) To assess the seizure frequency according to the sub type of stroke.

(4) To evaluate seizure frequency according to the site of lesion on neuroimaging.

(5) To correlate EEG finding with clinical profile of stroke seizures.

MATERIALS AND METHODOLOGY

STUDY DESIGN:-

The study is a prospective nonrandomized study done in tertiary care hospital based on clinical observations.

The study was carried out at Government Stanley Medical College Hospital, Chennai. Patients admitted in intensive care units, medical and neuromedical wards were enrolled from November 1st 2009 to December 31st 2010.

On admission all patients were examined in detail. These patients were investigated with relevant biochemical investigations and hemogram, CT scan brain plain(contrast given if needed), electroencephalography (EEG). electro cardiogram (ECG), 2D-ECHO and Doppler studies of neck vessels.

Other relevant investigations like MRI/MRA and CSF studies were done few patients when indicated clinically.

Patients were treated according to prevailing standards and were followed at regular interval.

Detailed analysis for data was performed and SPSS for windows version 10.0 was used for statistical analysis. Descriptive statistics was used for demographic profile. Cross tabulation, paired sample t test, and ANOVA test were used for comparison between groups for significance and p-value of <0.05 was considered as statistically significant.

INCLUSION CRIETERIA:-

(1) Patients presenting with recent onset of stroke

(2) Age above 18 years.

(3) Males and Females irrespective of race and religion

(4) Neurological deficit lasting > 24 hrs.

(4) CT scans showing normal study/infarction and/or cerebral hemorrhage

EXCLUSION CRITERIA:-

(1) Already diagnosed and undergoing treatment for epilepsy.

(2) Identifiable metabolic/systemic disorder that is known to cause seizures. (i.e, hyponatremia, severe hypo/hyper glycemia).

(3) Patient having transient ischemic attacks (TIA).

(4) CT Scan showing features suggestive of venous infarctions, subarachnoid hemorrhage, aneurysm, or arteriovenous malformation.

(5) Imaging showing identifiable non vascular causes of seizures like granuloma, tumors were excluded.

RESULTS AND ANALYSIS

Total of 164 patients were enrolled but 34 patients were excluded because lack of follow-up or essential investigation final analysis was performed on 130 patients.

There were 87 (67%) males and remaining were females (Figure 1), in age range of 25-75 years (mean: 54.8 ± 13.1 years). In seizures group mean age was 54.6 years and in seizure free group mean age was 54.8 years (p=0.945).

Altered sensorium was seen more frequently in seizure group (50.0%), than in seizure free group (37.9%), but difference was not statistically significant (p=0.377). No significant difference was seen between two groups regarding age, sex, occupation, time since onset, and side of weakness.

Seizures were observed in 14 (10.8%) patients during mean follow up period of about 136 days. Most of them had focal motor seizures. All patients with focal seizures had seizures contralateral to the side of lesion on CT scan. In focal seizure group 4 patients (33.33%) had left focal seizures and 8 patients (66.66%) had right focal seizures. Five patients had focal motor onset with secondarily generalized seizures. 12 Patients had seizures within 14 days of stroke (early onset seizures), and 2 patients had seizures after 14 days (late onset seizures) (Figure. 2). In early onset seizure group 8 patients had seizures within 48 hours of onset of stroke and remaining 4 patients had seizures between 48 hours and 14 days.

Table 1: Clinical profile (n=130)

Parameter	Number (%)
Males	87 (67%)
Females	43 (33%)
Age (Years)	54.8 ± 13.1
Time since onset (in days)	2.22 ± 1.63
Headache	23 (17.7%)
Weakness (History) ➢ Monoperesis ➢ Hemiperesis ➢ Quadriperesis	116 (89.2%) 5 (3.8%) 100 (76.9%) 11 (8.5%)
Altered Sensorium	51 (39.2%)
Seizures	14 (10.8%)
Type of Seizures * ➢ Focal ➢ Generalised	 12 * 2
Onset of Seizures ➢ Early (<14 days) ➢ Late (> 14 days)	 12 2

*5 patients had focal motor seizures with secondary generalisation.

In late onset seizures group one patient had one episode of GTCS six months after stroke, while another patient had three episodes of GTCS seven months after stroke. In early onset seizures group two patients presented with epilepsia partialia continua. Remaining 10 patients had 1-3 episodes of focal seizures. Only two patients had recurrent focal motor seizures up to one year of follow up.

Table 2: Risk Factors (n=130).

Risk Factors	Number (%)
Hypertension	45 (34.6%)
Diabetes mellitus	25 (19.2%)
Smoking	58 (44.6%)
Alcoholism	47 (36.27%)
Past H/O IHD/MI	6 (4.6%)
Atrial fibrillation	3 (2.3%)
Past Stroke	7 (5.4%)
Past TIA	8 (6.2%)
Family H/O Stroke	3 (2.3%)

Risk factors were not significantly different in patients with and without seizures. About 70% of the patients were found to have hypertension at the time of admission.

Table 3: Clinical parameters on Examination.

Parameters	**Total mean ± SD**	**Seizure group(n=14)**	**Seizure Free (n=116)**	**p-value**
BP(mmHg) - systolic - diastolic	155.3 ± 32.4 92.4 ± 16.7	147.1 ± 30.5 87.3 ± 14.9	156.3 ± 32.6 93.0 ±16.8	NS
GCS Score	12.1 ± 3.4	11.1 ± 2.9	12.2 ± 3.48	NS
MRS	3.5 ± 1.4	3.9 ± 1.3	3.4 ± 1.4	NS

On neurological examination 69 patients (53%) were fully conscious. Remaining patients were having varying degree of altered sensorium. 34 Patients were drowsy but arousable, 16 were localizing to painful stimuli, 5 patients were decerebrating and six patients did not have any response. Mean modified Rankin Scale score was 3.9 ± 1.3 in seizure group, while in seizure free group it was 3.4 ± 1.4 (p=0.19) weakness was detected in 113 patients (86.9%) at time of presentation. Out of these 32 patients (24.6%) had left sided weakness, 68 (52.3%) had right sided weakness, and 14 (10.0%) had quadriperesis. In 100 patients with unilateral weakness monoperesis was seen in 4% and hemiperesis in 96% patients.

Mean hemoglobin level was lower in patient with seizures as compared to seizure free group, and was statistically significant (p=0.006). There was a trend for higher fasting or random blood sugar in patients with seizures. Total cholestrol, HDL cholestrol, and LDL cholestrol were lower in patients with seizures as compared to seizure free group, but it was significant only in case of LDL cholestrol (p=0.047). Triglycerides, VLDL cholestrol, and electrolytes were not significantly different in two groups.

Patients were followed up for a mean period of 135.95 $\pm$ 145.2 days (range 1-540 days). In seizure group mean follow up was 138.7 $\pm$ 147.7 days, while in seizure free group mean follow up was 135.6 ± 145.5 days. The difference was not statistically significant (p=0.669). Patients who survived have mean follow up of 153.0 $\pm$ 146.5 days and in death group it was 29.7 $\pm$ 76.6 days.

Table 4: Hematological and Biochemical parameters.

Parameter (n)	**Total mean ± SD (Range)**	**Seizure group (n-14)**	**Seizure free group (n-116)**	**Significance (ANOVA)**
Hemoglobin in gm% (108)	12.61 ± 2.25 (5.8 – 17.6)	10.87 ± 2.35	12.81 ± 2.17	0.0006
Fasting/random Blood sugar in mg% (130)	121.98 ± 60.35 (59-444)	128.64 ± 36.7	12.17 ± 62.67	0.663
Total cholestrol in mg% (93)	181.99 ± 6.25 (92-292)	163.25 ± 36.70	183.75 ± 44.52	0.233
LDL Cholestrol in mg% (93)	106.64 ± 39.26 (14-210)	79.0 ± 39.3	110.09 ± 38.20	0.047
HDL Cholestrol in mg% (93)	40.10 ± 13.35 (18-80)	34.80 ± 10.50	40.75 ± 13.59	0.269
Triglycerides In mg% (93)	155.08 ± 70.64 (43-486)	158.63 ± 59.18	154.74 ± 71.92	0.883
VLDL Cholestrol in mg% (93)	31.14. ± 14.23 (9-97)	31.75 ± 11.72	31.08 ± 14.51	0.90
Serum Sodium in MEq/L (130)	139.22 ± 4.52 (127-150)	138.86 ± 5.38	139.86 ± 4.43	0.755
Serum potassium in MEq/L (130)	4.05 ± 0.54 (2.1-5.3)	4.09 ± 0.55	4.04 ± 0.54	0.764

Good outcome on follow up (defined as modified Rankin Score of 0-3) was seen in 92 patients (70.8%) and poor outcome (defined as mRS 4-6) in 38 patients (29.27%). In patient with seizures, good outcome was seen in 64.3% of patients while in seizure free group good outcome was seen in 71.6% of

patients. Though there was a trend for better outcome in seizures free group, it was not significant (p=0.549). Total 18 patients died during follow up. In seizures free group 14 patients (12.1%) died, while in seizure group 4 patients (28.6%) died. The difference was not statistically significant (p=0.0621). Sixteen patients died within one week of onset of stroke, while 2 patients died after six months.

Neuro Imaging: Computed Tomography (CT) scans of head were either normal or showed features of infarction in 108 patients (83.1%), while hemorrhage was seen in 22 patients (16.9%). Out of 116 patients free of seizures 96 (82.8%) showed normal CT scan or infarction, and 20 (17.2%) showed hemorrhage. In 14 patients with seizures 12 patients (85.7%) had normal or infarction on CT Scan and only 2 patients (14.3%) had features of hemorrhage (Figure. 4).

Table 5: Type of lesion on CT Scan.

CT Lesion	Total n=130	Seizure Group n=14	Seizure free Group n=116.	Significance
Infarct or Normal	108 (83.1%)	12 (85.7%)	96 (82.8%)	Not Significant
Hemorrhage	22 (16.9%)	2 (14.3%)	20 (17.2%)	Not Significant

Cortical lesions were seen in 26 patients (20%), sub cortical lesions in 47 patients (36.2%), and both cortical and subcortical lesions (large lesions) in 39 patients (30%). CT Scan head were normal in 18 patients (13.8%). In Patients with post stroke seizures cortical lesion were seen in 2 patients, subcortical lesion in 4 patients, and both cortical-subcortical (large lesions) in 6 patients. 2 patients in post stroke seizures group had normal CT Scan. Larger lesions were seen more commonly in seizure group as compared to patients without seizures, but it was not significant statistically (Figure. 5).

Table 6: Site of lesion in CT Scan

Site of Lesion	Total (130)	Seizure Group(14)	Seizure free (116)
Cortical	26 (20%)	2 (14.29%)	24 (20.69%)
Subcortical	47 (36.2%)	4 (28.5%)	43 (37.07%)
Both Cortical & Sub Cortical	39 (30.0%)	6 (42.86%)	33 (28.45%)
Not applicable	18 (13.85%)	2 (14.29%)	16 (13.79%)

92 patients (70.8%) had lesions in middle cerebral artery (MCA) territory, one patient (0.8%) in anterior cerebral artery (ACA), 8 patients in internal carotid artery territory (both MCA and ACA), and 16 patients (10%) had lesions in vertebrobasilar circulation. Right sided lesions were seen in 36 (27.7%), Left sided in 61 (46.91), and bilateral lesions in 21 patients (16.2%). CT Scan was normal in 12 patients. Difference in site of Lesion, Vascular territory involvement and side of lesion were not significant in patients with and without seizures. Seizures occurred in 2 of 22 patients with hemorrhagic stroke (9.1%) and in 12 of 108 patients with ischemic stroke (11.1%).

Electroencephalography (EEG) was performed in 43 patients (33.1%). In 87 patients EEG was not performed due to technical reasons. EEG was

normal in 17 patients, showed diffuse background slowing in 14 patients, hemispheric slowing in 11 patients and periodic lateralized epileptiform discharges (PLEDS) in one patient (Table 7).

Table 7: EEG Profile of study population

EEG Type	**Total (43)**	**Seizure Group (10)**	**Seizure free group (33)**
Normal	17 (39.5%)	3 (30%)	14 (42.4%)
Diffuse Slowing	14 (32.6%)	4 (40%)	10 (30.3%)
Hemispheric Slowing	11 (25.6%)	2 (20%)	9 (27.3%)
PLEDS	1 (2.3%)	1 (10%)	0 (0.0%)

Seizure and EEG changes did not have any significant association, on analysis for categorical variables. EEG abnormalities were seen more frequently in seizure group than in seizure free group. Only one patient with seizure showed PLEDS while none in seizure free group showed PLEDS. Correlation of EEG with CT Scan and other parameters was not possible due to small number in each group.

Electrocardiography (ECG) was performed in 126 patients (97%). ECG was normal in 84 patients (64.6%) and abnormal in remaining. Predominant

abnormality was left ventricular hypertrophy, three patients showed atrial fibrillation.

2D-echocardiography was done in 81 patients (62.3%). It detected abnormalities in 34 patients. Out of 34 patients with abnormal 2D-ECHO, 4 patients had rheumatic valvular heart disease, 16 patients had features of concentric left ventricular hypertrophy secondary to hypertension, 8 had ischemic heart disease with segmental hypokinesia and 6 patients showed sclerotic aortic valve.

Doppler neck vessels were performed in 88 patients (67.7%). Doppler studies were normal in 52 patients and abnormal in 36 patients. Out 36 patients with abnormality Doppler showed diffuse atheromatous changes but without hemodynamic compromise in 20 patients, hemodynamically significant stenosis of extracranial vessels were seen in 8 patients, near total occlusion of right and left internal carotid artery were seen in one patient each. Bilateral vertebral arteries (VA) were not visualized in 4 patients, right and left vertebral arteries were not seen in one patient each.

Figure.1 CT scan (plain) of a 55 years male having recurrent right focal motor seizures up to one year of follow up despite significant improvement in motor power.

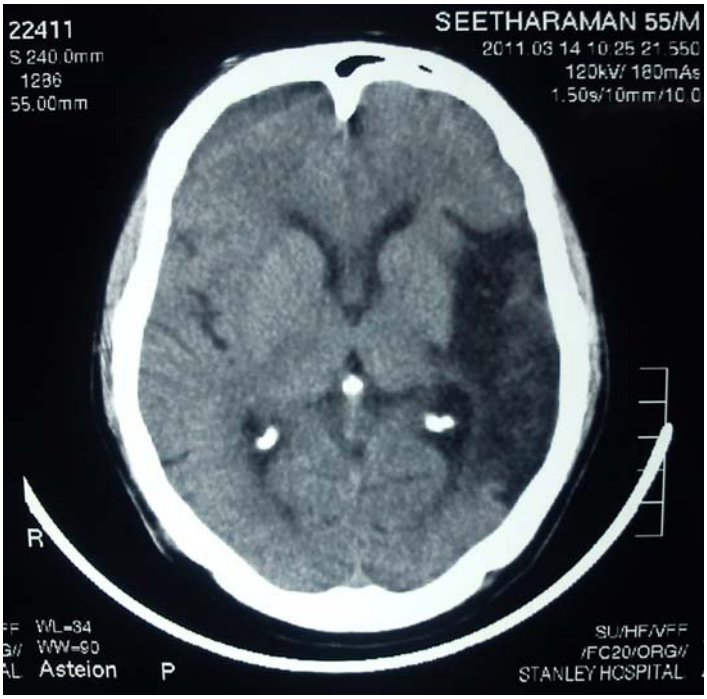
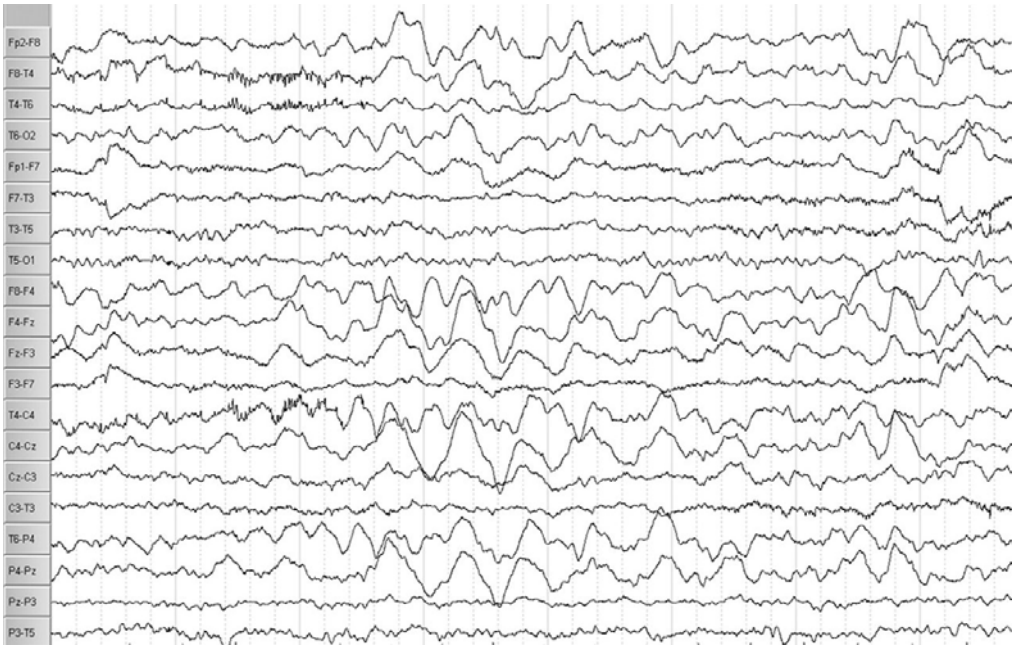


Figure.2 CT Scan (plain) of a 35 years old patient having right hemiplegia with global aphasia with seizures.

Figure.3 CT Scan (plain) of a 46 years old patient having intra cerebral hemorrhage with seizures.

Figure.4 EEG of a patient with large right MCA infarct having left focal motor seizures, showing right hemispheric slowing.



DISCUSSION

The age and other demographic profile were comparable to previous studies. Common risk factors for stroke are diabetes mellitus, hypertension, smoking and alcoholism. In our study the most common risk factor was smoking (44.6%), followed by alcoholism (36.2%). Hypertension and diabetes Mellitus were seen in 34.6% and 19.2% respectively. Earlier population based study by Banerjee TK et al[1] from Calcutta, reported hypertension as the most common risk factor. Reason for lower prevalence of hypertension and diabetes is not clear; one explanation may be that patients had undetected hypertension because at the time of admission about 70% of patients had hypertension.

Ischemic stroke is the most common sub type of stroke accounting for about 90% of all cases. In this study ischemic stroke was seen in about 83% and hemorrhagic stroke in 17%. The proportion of hemorrhagic stroke is higher than described in most western studies. Bladin CF et al[4] reported that 13.9% had hemorrhagic stroke. Kotila M et al[11] reported 10% incidence of hemorrhagic stroke out of 200 stroke patients. But Banerjee TK et al[1] found higher prevalence of hemorrhage in a population based survey in Eastern India. This may reflect the general tendency for higher prevalence of hemorrhagic stroke in Asian countries as compared to western countries. The present study was a hospitals based study so bias cannot be excluded.

There is no consensus regarding exact prevalence and predictors of post stroke seizures. Seizures usually occur in about 8-10% of stroke patients. Seizures were observed in 14 (10.8%) out of 130 patients in our study. Bladin CF et al[4] found post stroke seizures in 8.9% patients. Sung CY et al[6] reported seizures in 4.6% of patients with intra cerebral hemorrhage. John Burn et al[7] reported that stroke patients have about on 11.5% risk single or recurrent seizures in the first 5 years after a stroke. In a study by Giroud M et al[9] seizures occurred in 5.4% of stroke patients. Lancman ME et al[15] reported incidence of 10% of post stroke seizures. In study by Heuts-Van REP et al[24] ten percent patients had seizures following a symptomatic supratentorial brain infarct. In a study by Kilpatrick CJ et al[26] seizures occurred in 4.4% patients. So EL et al[29] in their study of 535 stroke patients reported early seizures (within 1 week) in 33 patients (6%), 78% of which occurred within first 24 hours after infarction. Twenty seven patients developed an initial late seizures (past 1 week), whereas 18 developed epilepsy. Weisberg et al[30] found that seizures occurred in 15% of patients with parenchymal brain hemorrhage. Majority of post stroke seizures are focal (partial) motor in type. Early onset seizures are more likely to be of generalized type. In present study 12 out of 14 patients had focal motor seizures. All patients had seizures contralateral to side of lesion on CT Scan. Twelve patients (87.7%) had early onset seizures and 2 patients had late onset seizures. All early onset seizures were generalized. In a study by Giroud M et al [9] seizures were the initial sign of stroke in 80 (89%) of 90 cases were usually single and partial. Kotila M et al (11) reported that 48% of 33

patients with seizures had generalized seizures and remaining had focal seizures. Sung CY et al[6] reported that seizure was the first manifestation of intra cerebral hemorrhage in 30% of patients. Gupta SR et al[8] in a retrospective study of 90 patients with post infarction seizures reported that 33% of post stroke seizures appeared early (within 2 weeks) and 90% of the 30 early seizures appeared within 24 hours. Early onset seizure were more likely to be partial (57% of 30); Late-onset seizures were more likely to be generalized (65% of 60). Giroud M et al[9] found that seizures were the initial sign of stroke in 80 (89%) of 90 cases with post stroke seizures. In series by Kotila M et al[11] 33/2000 consecutive stroke patients had seizures. Of these 15% had their first seizures within 2 weeks after stroke, and 55% developed epilepsy in 6 months. 48% of the patients had generalized seizures. Lancman ME et al[15] reported that 10% stroke patients had seizures. About 55% patients had early onset seizures (<one month), and remaining had late onset seizures. Kilpatrick CJ et al[26] reported that post stroke seizures generally occurred with 48 hours of stroke onset, were usually single, partial in nature. So EL et al[29] in their study reported that 33 patients (6%) developed early seizures (within 1 week), 78% of which occurred within the first 24 hours after infarction. Twenty seven patients developed an initial late seizure (past 1 week), whereas 18 developed epilepsy (recurrent late seizures).

Post stroke seizures are reported more frequently in patients having severe deficit, acute confusional state and various neurological and medical

complications. In present study history of altered sensorium was more frequent in post stroke group. Arboix A et al[17] reported that advanced age, confusional syndrome, hemorrhagic stroke, large lesions, involvement of parietal and temporal lobes and occurrence of neurologic and medical complications were significantly more frequent in seizure patients than in seizure free group.

Seizures are more commonly seen in hemorrhagic stroke as compared to ischemic stroke. In our study seizures occurred in 2 out of 22 patients with hemorrhagic stroke (9.1%) and in 12 of 108 (11.1%) patients with ischemic stroke. Bladin CF et al[4] found seizures in 10.6% of patients with hemorrhagic and 8.6% of patients with ischemic stroke. John Burn et al[7] described that patients with more severe strokes or hemorrhagic strokes are at higher risk for post stroke seizures. In a study by Giroud M et al[9] seizures occurred in 90/1640 patients (5.4%) including 36 (4.4%) of 814 with infarct due to atheroma, 21 (16.6%) of 126 with infarct due to cardiogenic embolus, 3 (1%) of 273 due to lacunar infarct, 5 (1.9%) of 259 due to transient ischaemic attack, 21 (16.2%) of 129 due to supra-tentorial haematoma, and 4 (16.6%) of 24 due to subarachnoid hemorrhage. In study by Kotila M et al[11] epilepsy developed in 17% of patients. The occurrence of epilepsy was 14% in ischaemic stroke, 15% in intracerebral hemorrhage and 35% in subarchnoid hemorrhage. Lancman ME et al (15) reported seizures in 22/219 stroke patients (10.04%). Seizures developed 13 of 183 patients with ischaemic stroke (7.1%) and 9 of 36 patients with hemorrhage stroke (25%). Kilpatrick CJ et al[26] reported seizures in

44/1000 patients (4.4%). 15.4% in lobar or extensive hemorrhage, 8.5% in subarachnoid hemorrhage, 6.5% cortical infarction, and 3.7% in hemispheric TIA had seizures.

Patients with cortical lesions or extensive lesions on imaging studies are more likely to have post stroke seizures. Lesion in anterior hemispheric distribution have higher incidence of post stroke seizures. In present study no significant association was seen with the site of lesion or vascular territory involved and post stroke seizures. Larger lesions were seen more commonly in seizure group as compared to patients without seizures, but it was not significant statistically. Sung CY et al[6] reported that incidence of seizure was 32% for lobar hematoma, 2% respectively for putaminal, thalamic and pontine hemorrhages and 1% for cerebellar hemorrhage. John Burn et al[7] described that risk of seizures after ischaemic stroke was substantial only in patients presenting with severe strokes due to total anterior circulation infarction. In study by Gupta SR et al (8) 67% patients with post stroke seizures had large infarctions on CT scan. Deep infarction on CT (cortical infarctions extending to sub-cortical structures) tended to cause recurrent seizures. Lancman ME et al[15] found that patients with cortical lesions and lesions involving more than one lobe are at higher risk of developing seizures. In study by Giroud M et al[23] 14.4% patients with post stroke seizures had a lenticulostriate infarct, diagnosed on CT scan, without an apparent ipsilateral cortical ischaemic lesion. MRI showed an associated Ipsilateral posterofrontal or anterotemporal cortical

ischemic lesion in 11 cases and SPECT showed decreased blood flow in the ipsilateral frontal area in all cases. Kilpatrick CJ et al[26] found that lacunar infarcts and deep hemorrhages were not associated with seizures. Arteriovenous malformation was a common cause of lobar hemorrhage with early seizures, but in cortical infarcts there was no association between seizure occurrence and pathogenesis. Labovitz DL et al[27] studied prevalence and predictors of early seizure. The frequency of early onset seizures by stroke sub type and location was deep infarct 0.6%, lobar infarct 5.9%, deep intracerebral hemorrhage 4.6%, lobar hematoma 14.3% and subarachnoid hemorrhage 8.0%. Weisberg LA et al[30] found that seizures were more frequent with lobar hemorrhages and uncommon with deep subcortical hemorrhages. Lobar hemorrhages in the frontal, parietal or temporal region were more commonly associated with seizures, whereas occipital hemorrahages were not.

Post stroke seizures can occur singly or may present as status epilepticus. In our study two patients (14.3%) with post stroke seizures presented with epilepsia partialia continua (status epilepticus). Sung CY et al[6] reported status epilepticus in 17% of post stroke seizures in intracerebral hemorrhage. In most patient seizures were focal and motor in nature. Gupta SR et al[8] in a retrospective study reported that status epilepticus occurred in only 8% patients. Rumbach L et al[13] reported that 159/3205 patients had post stroke seizures. Status epilepticus was seen in 31 patients (19%). In 17 patients, status epilepticus was the first epileptic symptom and in 4 patients, stroke

began with status epilepticus. In the 14 remaining patients, status epilepticus occurred after one or more seizures. Labovitz DL et al[27] reported status epilepticus in more than one quarter of patient with early onset seizures. Velioglu S et al[36] reported that 17 out of 180 patients (9%) with post stroke seizures had status epilepticus. Status epilepticus occurred more frequently among patients with a higher disability rating (Rankin Scale >3). Early onset status epilepticus was associated with a higher risk for recurrence ($p = 0.003$) and mortality rate ($p=0.04$).

Post stroke seizures are common but development of recurrent seizures or epilepsy is rare. In present study only two out of 14 (14.28%) patients with post stroke seizures had epilepsy. Sung CY et al[6] had reported epilepsy in 2.5% of patients with intracerebral hematoma. Bladin CF et al[4] reported recurrent seizures (epilepsy) in 2.5% of 1897 patients with stroke. Kilpatrick CJ et al[10] assessed the incidence of late onset seizures in 31 patients with early seizures complicating acute stroke. Ten (32%) of 31 patients with early seizures had late seizures during a mean follow up period of 26 months. Only three (10%) of 31 patients without early seizures had late seizures during the follow up period of 28 months, a significantly lower incidence than in patients with early seizures. Kotila M et al[11] in a retrospective study found epilepsy in 17% patients of stroke. The incidence of epilepsy was 14% in ischaemic stroke, 15% in parenchymal hematoma and 35% in subarachnoid hemorrhage.

There was significant difference in mean hemoglobin level in patients with and without seizures. Patients with seizures had lower hemoglobin (p=0.006). No other studies are available on effect of hematocrit on seizure frequency.

Higher cholesterol level is reported to have protective effect against post stroke seizures or prodromal seizures. Total cholesterol, HDL cholesterol and LDL cholesterol (p=0.047) were lower in patients with seizures as compared to seizures free patients. G Devuyst et al[16] found that serum cholesterol levels were lower in patients with prodromal or early onset post stroke seizures. Mortality and functional out come at discharge were not influenced. Mechanism of protective role of hypercholesterolemia for post stroke seizures is still not clear.

In our study post stroke seizures and electroencephalographic changes did not show any significant association. EEG abnormalities were seen more commonly in seizure group as compared to patients without seizures. Gupta SR et al[8] in retrospectively study of 90 patients with post infarction seizures found that most common EEG abnormality was focal slowing (61%), but recurrent seizures occurred in 100% of four patients with periodic lateralized epilepticform discharges and in 75% of the eight patients with diffuse slowing. Verma NP et al[32] described contralateral epileptiform transients (CETS) in EEG in cases of stroke. These discharges are unassociated with clinical seizure and appear to last longer than PLEDS or BIPLEDS. Sometimes, they may point

to a contralateral cerebral and / or cardiac pathology. In our study CETS were not seen. Gilmore PC[33] et al reported that stroke is the most frequent etiologic factor for focal delta activity. Tumors occurred less often. Convulsions were the most frequent cause of focal delta activity with a normal scan.

Post stroke seizures are known to cause increased morbidity and mortality in stroke patients. In present study there was trend for poorer out come and higher mortality in post stroke seizure group as compared to seizure free group. Paolucci S et al[19] studied the impact of seizures on the results of rehabilitation treatment. They evaluated the impact of post stroke seizures on both efficiency and effectiveness of rehabilitation and length of stay. No significant association was found between post stroke seizures and results of rehabilitation. Sung CY et al[6] reported that 34% patients with early seizures died within three months of seizures whereas only 12% with late seizures died within the same period. Reith J et al[12] in their study found that early onset seizures per se were not related to mortality. Surprisingly, early onset seizures predicted a better out come. Arboix et al[17] reported overall in-hospital mortality rate was 33.3% in the seizure group and 14.2% in the non seizure group ($p=0.02$). Seizures at the onset of a first ever stroke are an independent prognostic factor for in hospital mortality. Bogousslavsky J et al[21] reported effect of seizures on stroke sequelae. The described 10 patients with post stroke partial epileptic seizures that were followed by persistent worsening of the previous neurologic deficit. Persistent worsening was associated with

longer seizures and longer partial seizures before generalization. In a study of Kilpatrick CJ et al[26] seizures were not associated with a higher mortality or worse functional out come. In a study by Labovitz DL et al[27] early onset seizures do not predict 30-day mortality.

In our study no significant association was found between seizures and extra cranial Doppler results. Earlier study by Cocito L et al[22] reported occurrence of epileptic seizures in 141 patients with angiographically proven carotid or middle cerebral artery occlusive disease. Epileptic seizures occurred sometime during the clinical course of the disease in 17.3% of carotid patients and in 10.8% of middle cerebral artery disease patients, being mainly represented by partial motor seizures. In the carotid group, epilepsy was the presenting symptom in 6.7% of patients, whereas no middle cerebral artery occlusive disease patient had seizures prior to the appearance of focal neurological deficit.

STUDY LIMITATIONS:-

This study is conducted in a tertiary care referral hospital. The sample size is small and lacks long term follow up, so bias cannot be excluded.

SUMMARY

- Out of 130 patients 67% were males and 33% were females.
- Mean age was 54.8 ± 13.1 years.
- Ischaemic stroke was seen in 108 patients (83%), while hemorrhagic stroke was seen in 22 patients (17%).
- Smoking was the most common risk factor.
- Post stroke seizures were seen in 14 (10.8%) out of 130 patients.
- 12 patients had early onset seizures and 2 patients had late onset seizures.
- In early onset seizures 8 patients had seizures with 48 hours of onset and remaining 4 patients had seizures between 48 hours and 14 days.
- Altered sensorium was most commonly seen in seizure group.
- In early onset seizures group all patients had focal seizures and late onset seizures were of generalized tonic clonic in type.
- No significant correlation was seen between seizure and CT scan findings.

- No significant correlation was seen between clinical profile and EEG changes.

- Post stroke seizures patients had significantly lower mean hemoglobin level (p=0.006).

- Post stroke seizures patients had significantly lower LDL Cholestrol (P <0.047).

- Patients with post stroke seizures had trend for poor outcome and increased mortality.

CONCLUSION

This prospective study has evaluated the clinical profile of seizures in cases of acute stroke.

Single seizure, shorter duration, in this study did not show any adverse outcome however the patients who had early onset recurrent seizures focal becoming generalized, lasting for prolonged duration were associated with poor outcome.

If the study is extended to a larger population and longer duration with appropriate monitoring of stroke and seizure cascades will be of immense help to the acute stroke patients with seizures.

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ABBREVIATIONS FOR MASTER CHART

PS	-	Presenting symptoms
SET	-	Seizure Type
TOS	-	Time of seizure
PMH	-	Past Medical History
CTS	-	Computerized Tomography Scan
EEG	-	Electro encephalography
ECG	-	Electro cardiography
ECHO	-	Echocardiography
SOL	-	Site of Lesion on Computerized Tomography Scan
M	-	Male
F	-	Female
RHP	-	Right Hemiperesis
LHP	-	Left Hemiperesis
AS	-	Altered sensorium
SE	-	Seizures
HA	-	Headache
RPS	-	Right partial seizures
LPS	-	Left partial seizures
EOS	-	Early onset seizures
LOS	-	Late onset seizures
HT	-	Hypertension
DM	-	Diabetes mellitus
IHD	-	Ischemic heart disease
AL	-	Alcoholism

SM - Smoking

INF - Infarct

ICH - Intracerebral Hemorrhage

NCT - Normal Computerized tomography

A - Abnormal

N - Normal

COR - Cortical

SC - Subcortical

Gender Distribution

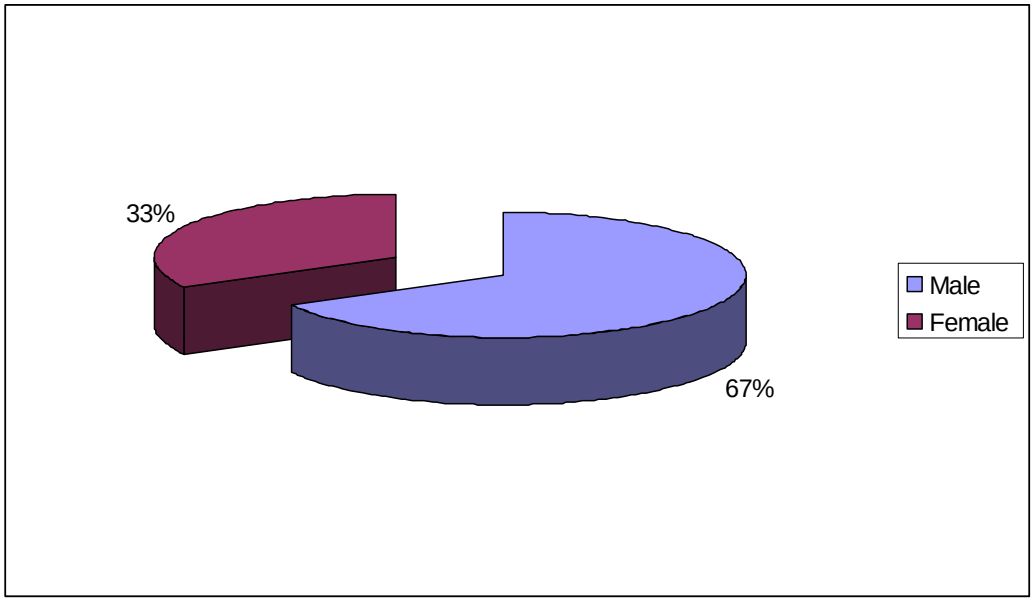

Figure. 1

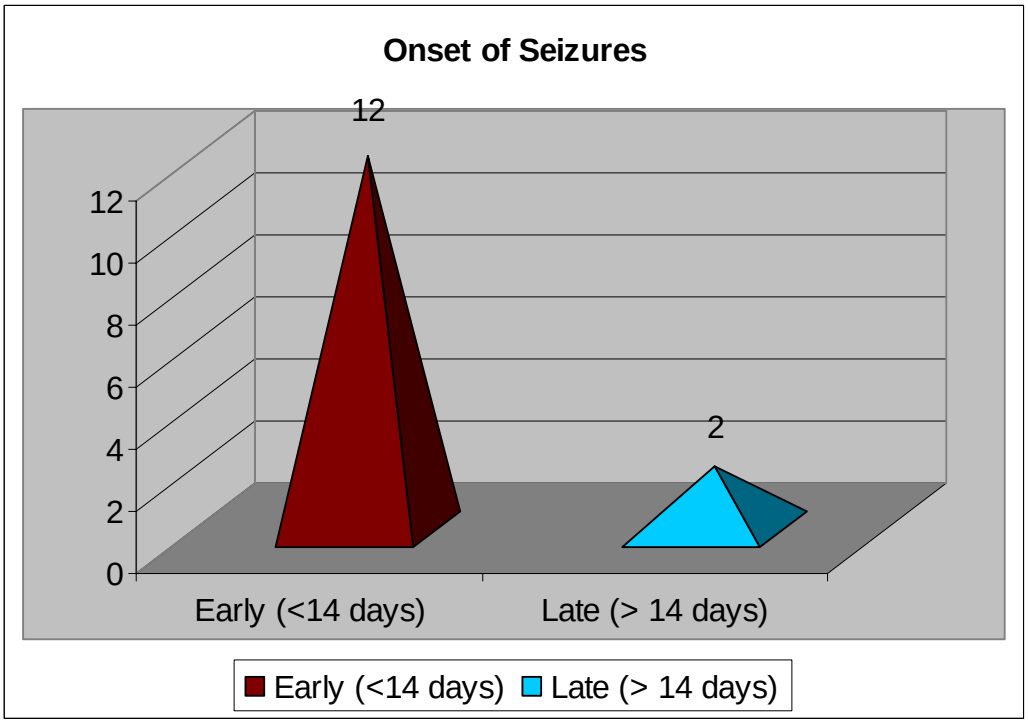

Figure. 2

Type of lesion on CT Scan

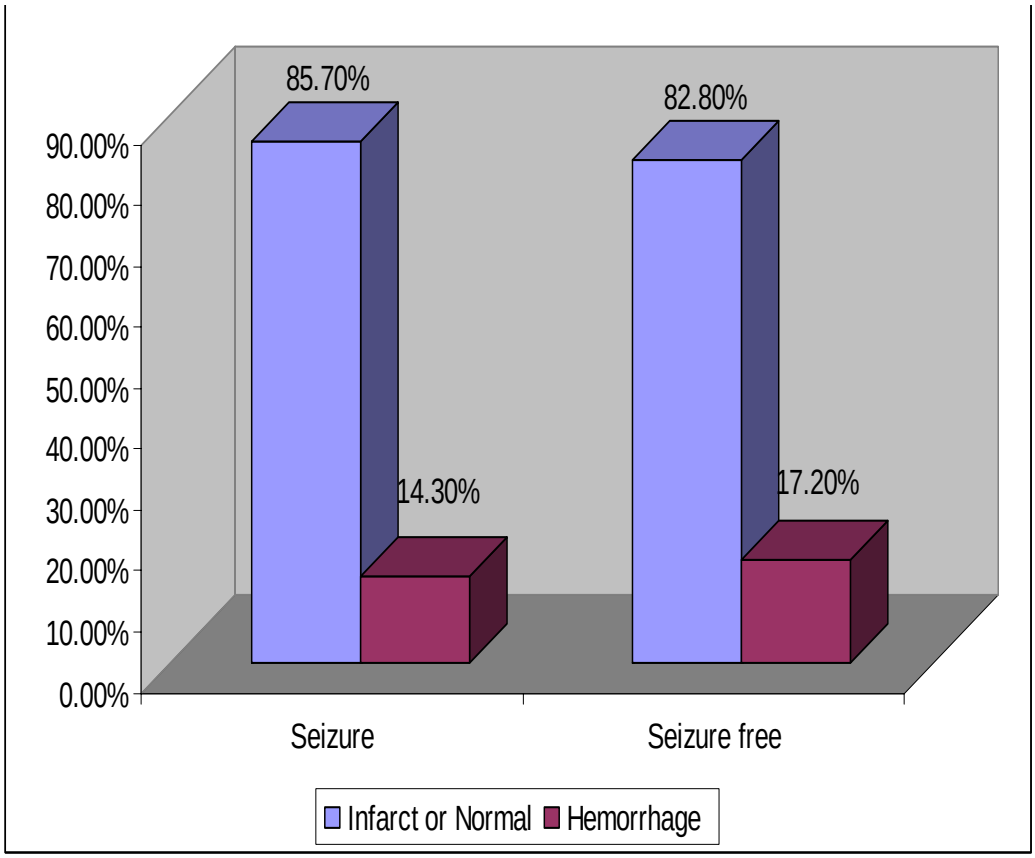

Figure. 4

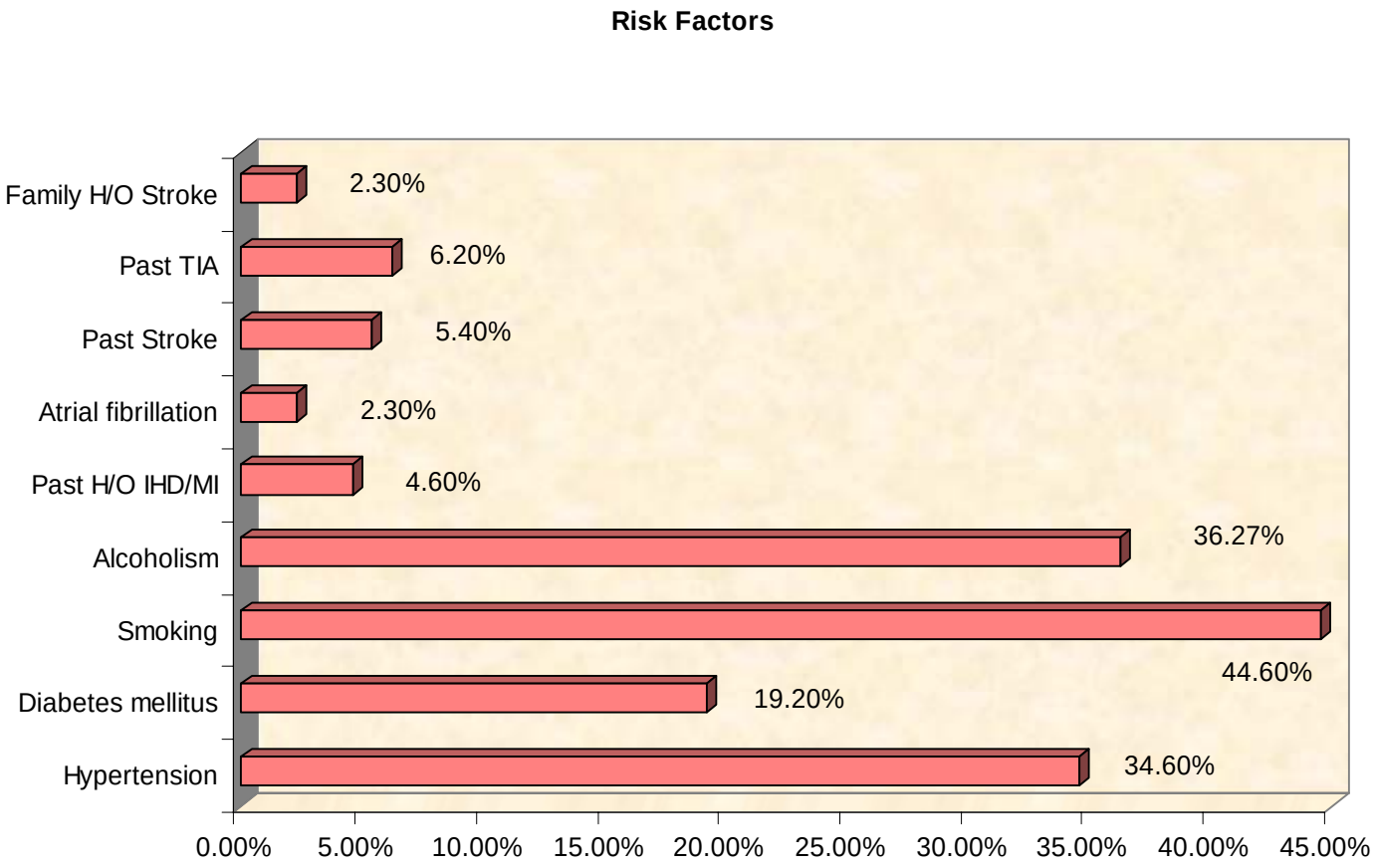
Risk Factors
Family H/O Stroke
2.30%
Past TIA
6.20%
Past Stroke
5.40%
Atrial fibrillation
2.30%
Past H/O IHD/MI
4.60%
Alcoholism
36.27%
Smoking
44.60%
Diabetes mellitus
19.20%
Hypertension
34.60%
0.00%
5.00%
10.00%
15.00%
20.00%
25.00%
30.00%
35.00%
40.00%
45.00%

Figure. 3

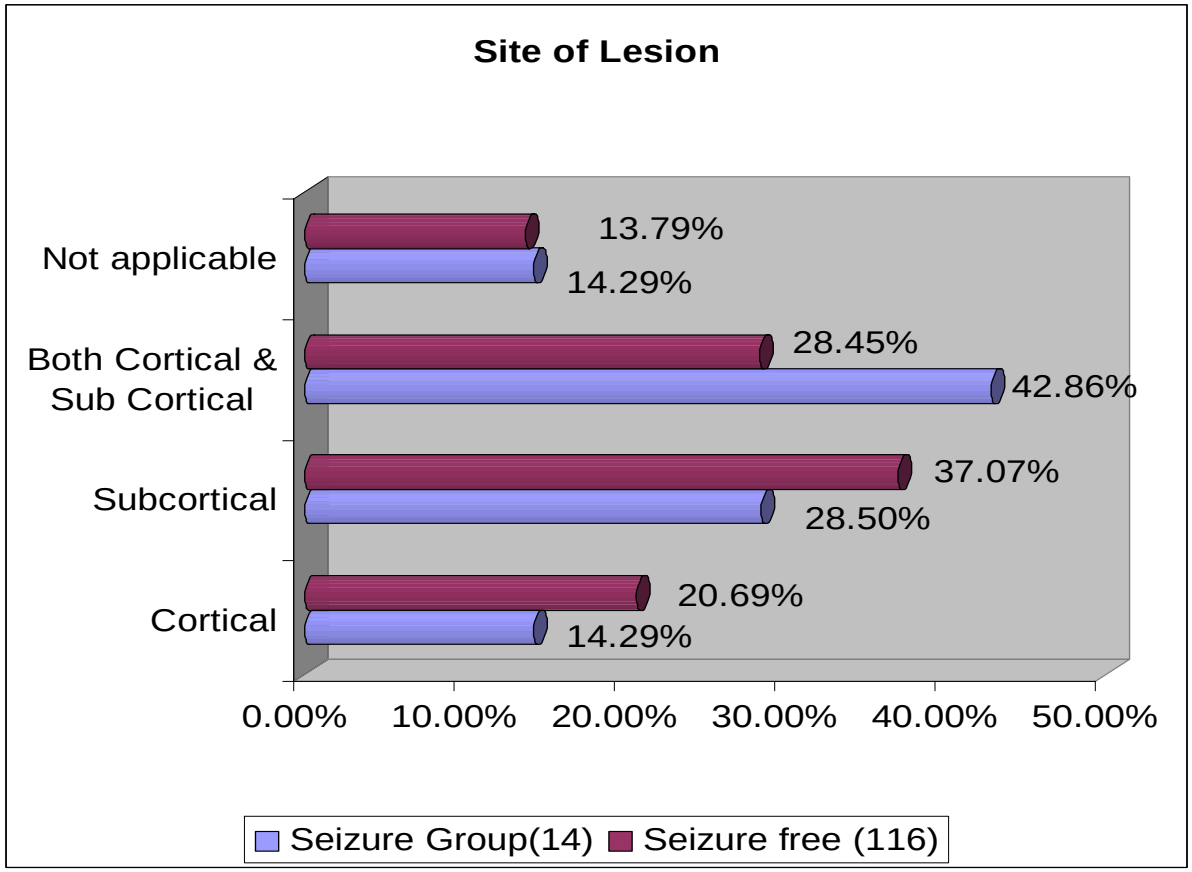
Site of Lesion
Not applicable
Both Cortical & Sub Cortical
Subcortical
Cortical
13.79%
14.29%
28.45%
42.86%
37.07%
28.50%
20.69%
14.29%
0.00%
10.00%
20.00%
30.00%
40.00%
50.00%
Seizure Group(14)
Seizure free (116)

Figure. 5

MASTER CHART

S. No.	Name	Age	Sex	PS	SET	TOS	PMH	CTS	EEG	ECG	ECHO	SOL
1.	Govindan	48	M	RHP	-	-	HT, DM, IHD	INF	A	A	A	COR
2.	Annammal	52	F	RHP	-	-	HT	ICH	-	N	N	SC
3.	Tamilselvan	56	M	LHP	-	-	AL, SM	NCT	N	N	N	NCT
4.	Anandan	58	M	AS, SE, RHP	RPS	EOS	HT, DM AL, SM	INF	A	A	A	COR & SC
5.	Krishnamoorthy	48	M	HA, RHP	-	-	SM	INF	-	A	-	SC
6.	Abdul Rahman	54	M	LHP	-	-	HT, AL, SM	ICH	-	N	N	SC
7.	Chellammal	70	F	AS, QP	-	-	IHD	INF	N	N	N	COR & SC
8.	Kathirvel	30	M	AS, RHP	-	-	AL, SM	INF	-	N	-	SC
9.	Velu	41	M	RHP	-	-	AL, SM	NCT	-	N	-	NCT
10.	Shanthi	43	F	AS, HA, RHP	-	-	HT	INF	-	A	A	COR & SC
11.	Elumalai	55	M	AS, RHP	-	-	-	INF	-	N	N	SC
12.	Dhatchayani	40	F	RHP	-	-	-	INF	-	A	N	SC
13.	Ravanan	58	M	AS, RHP	-	-	AL, SM	ICH	-	N	N	COR & SC
14.	Varadhan	64	M	LHP	-	-	HT, DM	INF	-	A	A	COR
15.	Ragupathy	46	M	HA, RHP	-	-	-	INF	-	N	-	SC
16.	Syed Hussain	48	M	RHP	-	-	SM	INF	N	N	-	COR & SC
17.	Radha	62	F	AS, SE, LHP	LPS	EOS	-	INF	A	N	N	COR
18.	Durai	36	M	RHP	-	-	AL, SM	NCT	-	N	-	NCT
19.	Padmaraj	48	M	HA, SE, AS, RHP	GTCS	LOS	AL, SM, HT, CVD	INF	A	A	A	COR & SC
20.	Sudakar	38	M	HA, RHP	-	-	-	INF	-	N	-	COR
21.	Seetha	64	F	QP	-	-	HT, CVD	ICH	N	A	A	COR
22.	Thangaraj	42	M	AS, LHP	-	-	DM	INF	-	N	N	SC
23.	Shankar	56	M	RHP	-	-	AL, SM, IHD	INF	-	N	-	COR & SC
24.	Murugan	46	M	RHP	-	-	HT, AL, SM	INF	A	A	A	COR
25.	Jamuna	48	F	AS, LHP	-	-	-	INF	-	N	N	SC
26.	Sudakar	42	M	RHP	-	-	SM	NCT	-	N	N	NCT

S. No.	Name	Age	Sex	PS	SET	TOS	PMH	CTS	EEG	ECG	ECHO	SOL
27.	Chandran	54	M	HA, RHP	-	-	-	INF	-	N	-	SC
28.	Iyyamperumal	62	M	AS, QP	-	-	HT, DM, AL, SM	ICH	-	A	A	COR & SC
29.	Thameem Bee	68	F	AS	-	-	-	INF	-	N	-	COR & SC
30.	Appasamy	50	M	LHP	-	-	HT	INF	N	A	A	SC
31.	Durai	46	M	SE, RHP	RPS	EOS	-	INF	A	N	N	COR & SC
32.	Pechiammal	50	F	LHP	-	-	-	INF	-	N	N	SC
33.	Suresh	38	M	HA, RHP	-	-	AL, SM	NCT	-	N	-	NCT
34.	Lakshmi	62	F	AS, RHP	-	-	HT	ICH	A	N	-	COR & SC
35.	Shajakhan	50	M	AS	-	-	DM, AL, SM	INF	N	A	A	COR
36.	Arunachalam	56	M	RHP	-	-	-	INF	A	N	N	COR
37.	Sivarajan	48	M	LHP	-	-	AL, SM	INF	-	-	-	SC
38.	Kamala	50	F	AS, RHP	-	-	-	INF	-	N	N	COR
39.	Subramani	40	M	HA, RHP	-	-	HT, DM, AL, SM	ICH	N	A	A	COR
40.	Ahamed Meeran	42	M	LHP	-	-	SM	INF	-	N	N	SC
41.	Kaliammal	56	F	AS, RHP	-	-	-	INF	-	N	N	COR & SC
42.	Viswanathan	44	M	AS, SE, RHP	RPS	EOS	DM	INF	A	A	A	COR & SC
43.	Arokkiyasamy	60	M	QP	-	-		NCT	-	N	-	NCT
44.	Kamatchi	70	F	LHP	-	-	HT, CVD	ICH	-	A	-	SC
45.	Paulraj	46	M	HA, AS, RHP	-	-	DM, SM, AL, CVD	INF	N	A	A	COR & SC
46.	Govindammal	54	F	AS	-	-	IHD	INF	-	N	-	COR & SC
47.	Kirubanithi	48	M	RHP	-	-	AL, SM	INF	-	N	-	SC
48.	Lakshmi	60	F	LHP	-	-	-	INF	-	N	N	SC
49.	Arumugam	55	M	RHP	-	-	AL, SM	INF	-	N	N	SC
50.	Mani	42	M	AS, RHP	-	-	HT	ICH	-	A	A	COR
51.	Rajakani	60	F	QP	-	-	HT, DM	INF	N	A	A	COR
52.	Chandramouli	52	M	AS, RHP	-	-	AL, SM	INF	A	N	-	COR
53.	Muthalagan	44	M	RHP	-	-	SM	NCT	-	N	-	NCT
54.	Poongavanam	40	F	RHP	-	-	HT	INF	-	N	-	SC
55.	Sriram	48	M	HA, AS	-	-	DM, AL, SM	INF	-	N	-	COR & SC

S. No.	Name	Age	Sex	PS	SET	TOS	PMH	CTS	EEG	ECG	ECHO	SOL
56.	Sulochana	51	F	AS, SE, LHP	LPS	EOS	HT	ICH	A	A	N	COR
57.	Shaul Hameed	50	M	AS	-	-	SM	INF	-	N	N	COR
58.	Rajendran	46	M	HA, LHP	-	-	HT, DM	INF	N	A	A	COR & SC
59.	Suseela	72	F	QP	-	-	-	INF	-	N	-	SC
60.	Seetharaman	55	M	SE, RHP	GTCS	LOS	AL, SM, CVD	INF	A	N	-	COR
61.	Rajamanickam	48	M	HA, AS	-	-	-	INF	-	N	N	COR
62.	Baby	54	F	RHP	-	-	HT	INF	-	A	N	SC
63.	Abdulla	60	M	LHP	-	-	-	INF	-	N	N	COR & SC
64.	Varadharaj	57	M	AS, SE, RHP	RPS	EOS	HT, DM, AL, SM	ICH	A	A	A	COR & SC
65.	Erusammal	64	F	AS	-	-	-	INF	-	N	-	COR
66.	Nagammal	5	F	LHP	-	-	-	NCT	N	N	-	NCT
67.	Arasan	62	M	QP	-	-	CVD	INF	-	N	N	SC
68.	Prakash	50	M	RHP	-	-	HT, DM, AL, SM	INF	-	A	N	SC
69.	Krishnaveni	38	F	HA, AS	-	-	HT	ICH	-	N	-	SC
70.	Munusami	40	M	LHP	-	-	HT, SM, AL	INF	-	A	A	COR & SC
71.	Jayaraman	60	M	AS, RHP	-	-	SM	NCT	-	N	N	NCT
72.	Ravi	47	M	RHP	-	-	AL, SM	INF	N	N	-	SC
73.	Saroja	44	F	LHP	-	-	-	INF	-	A	-	SC
74.	Ramachandran	56	M	RHP	-	-	AL, SM	NCT	-	N	-	NCT
75	Manavalan	60	M	HA, AS	-	-	HT	INF	A	N	N	COR & SC
76.	Majith	41	M	RHP	-	-	HT	ICH	N	A	A	SC
77.	Adilakshmi	68	F	LHP	-	-	DM	INF	-	N	N	COR & SC
78.	Vijayan	50	M	AS, SE, LHP	LPS	EOS	HT, SM, AL	INF	A	A	A	COR
79.	Loganathan	55	M	RHP	-	-	HT	INF	-	N	-	SC
80.	Muniammal	72	F	RHP	-	-	HT CVD	ICH	-	N	N	COR & SC
81.	Boominathan	58	M	AS, QP	-	-	DM, AL, SM	INF	-	A	A	COR & SC
82.	Manickavelan	52	M	AS, HA	-	-	IHD	INF	-	A	A	COR
83.	Jaya	30	F	RHP	-	-	-	NCT	-	N	-	NCT
84.	Palani	58	M	RHP	-	-	AL, SM	INF	N	N	-	SC

S. No.	Name	Age	Sex	PS	SET	TOS	PMH	CTS	EEG	ECG	ECHO	SOL
85.	Vijaya	49	F	LHP	-	-	-	INF	-	N	N	SC
86.	Babu	37	M	RHP	-	-	AL, SM	NCT	-	N	N	NCT
87.	Parthiban	46	M	RHP	-	-	HT, SM	ICH	-	A	A	COR & SC
88.	Gowri	42	F	RHP	-	-	HT, DM	INF	N	A	A	SC
89.	Anbazhagan	55	M	LP	-	-	-	INF	-	N	-	SC
90.	Subramani	56	M	AS, SE, RHP	RPS	EOS	HT, AL, SM	INF	A	A	A	COR & SC
91.	Sudamani	60	F	RHP	-	-	HT	INF	-	A	-	COR
92.	Nagappan	65	M	AS, QP	-	-	DM	NCT	-	N	-	NCT
93.	Madurai	45	M	HA, AS	-	-	AL, SM	INF	N	N	N	COR
94.	Anusuya	38	F	RHP	-	-	HT	ICH	-	N	N	SC
95.	Arokkiyasamy	56	M	LHP	-	-	AL, SM	INF	A	N	-	COR & SC
96.	Palayam	55	F	RHP	-	-	-	INF	-	N	-	COR
97.	Rajagobal	60	M	AS, QP	-	-	-	NCT	-	N	N	NCT
98.	Eswaran	44	M	AS, SE, RHP	RPS	EOS	DM, SM	INF	A	A	A	COR & SC
99.	Ruckmani	50	F	RHP	-	-	DM	ICH	-	N	N	SC
100.	Elumalai	54	M	AS, QP	-	-	HT, DM	INF	-	A	A	COR
101.	Sasikumar	48	M	LHP	-	-	HT, AL, SM	INF	N	A	A	SC
102.	Nagarathinam	42	F	AS, RHP	-	-	-	INF	-	N	-	COR & SC
103.	Kothandan	48	M	RHP	-	-	AL, SM	ICH	-	N	-	SC
104.	Selvam	36	M	LHP	-	-	AL, SM	INF	A	N	N	COR & SC
105.	Velammal	72	F	HA, AS	-	-	HT	INF	-	A	-	COR
106.	Elangovan	56	M	RHP	-	-	HT, AL, SM	INF	-	N	N	SC
107.	Veerabathiran	45	M	LHP	-	-	AL, SM	INF	-	N	N	COR
108.	Kala	46	F	RHP	-	-	HT	ICH	-	N	N	COR & SC
109.	Raja	30	M	RHP	-	-	SM	INF	-	N	-	SC
110.	Thirumalai	52	M	LHP	-	-	HT, AL, SM	INF	A	A	A	COR & SC
111.	Chithra	38	F	HA, AS	-	-	-	INF	-	N	-	COR & SC
112.	Deenan	47	M	SE, RHP	RPS	EOS	-	ICH	A	A	A	COR & SC
113.	Dharmarajan	60	M	RHP	-	-	HT, DM	NCT	-	N	N	NCT

S. No.	Name	Age	Sex	PS	SET	TOS	PMH	CTS	EEG	ECG	ECHO	SOL
114.	Mallika	50	F	LHP	-	-	HT	INF	-	N	-	SC
115.	Murugesh	49	M	RHP	-	-	SM	INF	-	N	N	SC
116.	Moorthy	42	M	HA, AS	-	-	AL, SM	INF	N	A	-	COR
117.	Muniammal	74	F	LHP	-	-	HT	INF	N	N	SC	SC
118.	Vinayagam	52	M	RHP	-	-	DM, AL, SM	INF	A	N	-	COR
119.	Rathinam	70	F	AS, HA	-	-	HT	INF	-	A	A	COR & SC
120.	Selvaraj	46	M	HP	-	-	AL, SM	ICH	N	N	N	SC
121.	Munusamy	55	M	SE, LHP	LPS	EOS	HT	INF	A	N	N	COR & SC
122.	Saraswathi	40	F	HA, AS	-	-	DM	INF	-	N	-	COR
123.	Sathiyamoorthy	57	M	RHP	-	-	AL, SM	NCT	-	N	-	NCT
124.	Ponraj	55	M	LHP	-	-	HT, CVD	INF	A	A	A	COR & SC
125.	Theresa	68	F	AS, HA	-	-	HT	ICH	N	N	N	SC
126.	Muthu	65	M	RHP	-	-	AL, SM	INF	-	N	-	SC
127.	Baskaran	43	M	RHP	-	-	AL, SM	NCT	-	N	N	NCT
128.	Kamatchi	48	F	AS, LHP	-	-	DM	INF	A	A	A	COR
129.	Narayanan	59	M	SE, RHP	RPS	EOS	HT	INF	A	A	A	COR & SC
130.	Vadivel	52	M	LHP	-	-	AL, SM	INF	-	N	N	SC

PROFORMA

Name :

Age :

Sex :

Neuro No./I.P. No. :

Educational Status :

Occupation :

Address :

Clinical Presentation :

Type of Stroke :
- Monoperesis
- Hemiperesis
- Quadriperesis

Type of Seizures :
- Generalized
- Focal

Time of Seizure after Stroke :

Past History :
- Hypertension
- Diabetes Mellitus
- Past H/O IHD/MI
- Past Stroke
- Past TIA

Alcoholism :

Smoking :

Family H/O Stroke :

General Examination :

- Built :
- Anemia :
- Jaundice :
- Cyanosis :
- Clubbling :
- JVP :
- Lymphadenopathy
- Pedal edema
- Neuro cutaneous markers

Pulse :
Blood Pressure :
Respiratory rate :

CNS :

- Higher Functions :
- Cranial Nerves :
- Spinomotor system :
  - Bulk :
  - Tone :
  - Power :
  - Co-ordination :
  - Involuntary Movements :

Reflexes :

- Superficial :
- Deep :

Sensory System :
Cerebellar signs :
Bladder & Bowel :
Spine & Cranium :
Meningeal Signs :

CVS :
RS :
Abdomen :

INVESTIGATIONS:

CBC :

Urine (R) :

RBS :

RFT :

Electrolytes :

Lipid Profile :

Chest X-ray :

ECG :

EEG :

CT Brain / MRI :

Echo :

Doppler study :
(Carotid & vertebral)

DIAGNOSIS

TREATMENT

OUTCOME